out, hemodynamics of coronary blood flow may be significantly different in a dilated failing heart compared with a normal heart, especially in the long term after cardiomyoplasty. We are currently using the Doppler guidewire system to evaluate long-term effects of cardiomyoplasty on coronary arterial blood flow in canine hearts. This information will resolve the contradictory findings and be useful in clarifying the mechanisms of augmentation by cardiomyoplasty.

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12/8/63334

The role of adenosine in University of Wisconsin solution

To the Editor:

The recent article by Lasley and Mentzer[1] investigating the role of adenosine in enhanced myocardial preservation with University of Wisconsin (UW) solution shows the important role of the compound for the protective properties of UW solution. Functional recovery as measured by rate-pressure product was significantly improved after 18 hours of cold storage with the inclusion of adenosine in UW solution. However, no difference was observed between UW solution without adenosine and St. Thomas' Hospital cardioplegic solution. The question arising is whether the inclusion of adenosine in St. Thomas' Hospital solution would have had the same beneficial effects on functional recovery and interstitial fluid purine levels. It can be concluded from the presented data that no difference exists between UW solution without adenosine and unmodified St. Thomas' Hospital solution and that the main component leading to improved preservation is adenosine.

Therefore, the conclusion drawn by the authors that the superior preservation with UW solution is a result of its intracellular-based electrolyte composition and inclusion of lactobionate, raffinose, and hydroxyethyl starch is misleading. We have shown a dose-dependent improvement of myocardial recovery with the addition of adenosine to St. Thomas' Hospital solution No. 2 in an in vivo baboon model after 3 hours of cardioplegic arrest.[2] Our results, consistent with those of other studies, showed no beneficial effect on myocardial adenosine triphosphate content, stressing the fact that the effect of adenosine on improving postischemic refunction seems to be independent of its effects as an adenine nucleotide precursor.

The assumption that the low-sodium, high-potassium formulation of UW solution could presumably retard cell swelling by reducing the electrochemical gradients of these ions may be correct; however, the high potassium concentration is possibly the cause of postischemic endothelial dysfunction as a result of endothelial damage or disturbance. Amrani and colleagues[3] reported a marked increase in coronary vascular resistance associated with impaired myocardial protection when using UW solution in an isolated rat heart model for 60 minutes of global ischemia at a moderately hypothermic temperature (20° C). Loss of protection with UW solution at temperatures of 20° C compared with St. Thomas' Hospital solution No. 2 seems to be correlated with the high potassium concentrations. Other groups have also reported impaired endothelium-dependent coronary responses after cold potassium cardioplegia.[4]

These findings show that the superior preservation with UW solution compared with other crystalloid preservation solutions is not uncontradicted. Another study from the Universities of Cape Town and Jerusalem found superior long-term preservation for 18 hours of cold storage with St. Thomas' Hospital solution No. 2 compared with UW solution in a paracorporeal porcine model.[5] Therefore, a study of a fourth group with St. Thomas' Hospital solution and inclusion of adenosine should be performed to substantiate the conclusions drawn by Lasley and Mentzer.

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12/8/61690

Reply to the Editor:

We thank Drs. Boehm and Reichart for their interest in our recent study on the role of adenosine in University of Wisconsin (UW) preservation solution.[1] In these studies we reported that omission of adenosine from UW solution markedly reduced the functional recovery of isolated perfused rabbit hearts after 18 hours of hypothermic (4° C) preservation. Functional recovery in hearts preserved with UW solution without adenosine was similar to that in hearts preserved with St. Thomas' Hospital solution. We concluded that adenosine plays a key role in the enhanced myocardial preservation observed with UW solution.

We agree with Drs. Boehm and Reichart that the question remains as to whether inclusion of adenosine in St. Thomas' Hospital solution would have produced the same functional recovery as in UW solution. Addition of adenosine to St. Thomas' Hospital solution would have likely produced the same interstitial fluid profile as seen with UW solution and improved the preservation, but, because the two solutions are so different in composition, it is difficult to say whether the functional recovery would have been equivalent to that of UW solution. However, the purpose of this study was to determine whether adenosine was a necessary component of UW solution, not to compare the preservation capabilities of the two different solutions. The St. Thomas' Hospital solution group was included in this study merely for comparison purposes.

The composition of UW solution is based on the complications associated with prolonged hypothermia and ischemia, but few studies have assessed which of these components play an integral role in preservation.[2] The results of this study indicate that adenosine is a key component of UW solution. In addition, Ko and colleagues[3] recently reported that reversal of the sodium-potassium composition or omission of hydroxyethyl starch resulted in less preservation of diastolic function after 6 hours preservation (1° to 2° C) of canine hearts. We agree that the effects of the high potassium concentration (in UW solution) on vascular/endothelial function must be studied further; however, in this study no evidence was found of more vascular dysfunction in hearts preserved with UW solution than in those preserved with St. Thomas' Hospital solution. Furthermore, in the study by Amrani and colleagues[4] cited by the respondents, the cold-storage temperature was much higher (20° C) than that for which the UW solution was designed (4° C).

In conclusion, on the basis of experimental results, it appears that UW solution may be superior to St. Thomas' Hospital solution for prolonged myocardial preservation because of the inclusion of adenosine and hydroxyethyl starch and its intracellular-based ionic composition. However, because clinical preservation times have remained in the 4 to 5 hour range for the past decade, it is evident that much work remains to be done in the area of myocardial preservation for heart transplantation.

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12/8/62941

Invited letter concerning: Biochemical and cellular characterization of cardiac valve tissue after cryopreservation or antibiotic preservation (J THORAC CARDIOVASC SURG 1994;108:63-7)

To the Editor:

The vexing question of whether aortic valve allografts survive longer when their donor cell population is viable at implantation has been debated now for more than 20 years. Truly conclusive evidence in favor of maintaining cell viability during the preparation and storage of grafts is still lacking. Most follow-up studies of so-called "viable" valve grafts have not attempted to establish the persistence of donor cells in the explanted valves or to define the histologic status of the grafts. A notable exception is the paper of O'Brien and colleagues,[1] who demonstrated persistent donor cells in one single case of the series they studied. Despite good cellularity, however, this graft did not exhibit normal leaflet architecture. Without adequate cytologic and histologic information on the "viable" graft at the end of its useful life, the possibility remains[2] that